1: Dev Genet 1999;24(3-4):299-308 Related Articles, Books, LinkOut

Peptides from the PKD repeats of polycystin, the PKD1 gene product, modulate pattern formation in the developing kidney.

van Adelsberg J.

Department of Medicine, Columbia University, New York, New York 10032, USA. jsv1@columbia.edu

Mutations in the PKD1 gene cause the majority of cases of autosomal dominant polycystic kidney disease. The PKD1 gene codes for a protein of unknown function, polycystin-1, that is predicted to be a receptor. Its large extracellular domain contains 16 copies of novel motif, the PKD repeat, that is likely to be a ligand binding domain based on its similarity to immunoglobulin domains. These observations suggested that soluble fragments of the extracellular domain of polycystin-1 could be used as competitive inhibitors of polycystin function in a suitable model system. Polycystin-1 is highly expressed in the ureteric bud and other branching epithelia during development and interacts with beta-catenin, a molecule known to play a role in branching morphogenesis. These data suggested that polycystin-1 might play a role in branching morphogenesis. I show here that peptides derived from the PKD repeats of polycystin-1 caused an asymmetric pattern of ureteric bud branching in cultured kidney rudiments. Treatment of kidney rudiments with experimental but not control peptides reduced both the number of ureteric bud branches and the number of nephrons. Experimental peptides produced significant morphogenetic effects at concentrations < or = 0.1 mM. These data suggest that polycystin-1 plays a role in branching morphogenesis by the ureteric bud.

PMID: 10322638 [PubMed - indexed for MEDLINE]

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The PKD1 gene product, "polycystin-1," is a tyrosine-phosphorylated protein that colocalizes with alpha2beta1-integrin in focal clusters in adherent renal epithelia.

Wilson PD, Geng L, Li X, Burrow CR.

Department of Medicine, Mount Sinai School of Medicine, New York, New York 10029, USA. pat.wilson@SMTPlink.mssm.edu

Mutations in the PKD1 gene are responsible for autosomal dominant polycystic kidney disease (ADPKD). Although PKD1 has been cloned and shown to be expressed at high levels in the fetal ureteric bud and ADPKD cystic epithelia in the human kidney, the function of its encoded protein, "polycystin-1" is unknown. In this study we used primary and immortalized human renal epithelial cell lines derived from normal fetal, adult, and ADPKD kidneys, that endogenously express PKD1, to study the biologic function of the polycystin-1 protein. ADPKD renal epithelial cells expressed high levels of polycystin-1 protein and showed increased adhesion to type I collagen by comparison with normal adult human renal epithelia that expressed little polycystin. Adherent ADPKD cells also expressed high levels of alpha2beta1-integrin and their attachment was inhibited by a functional monoclonal antibody to alpha2-integrin. Double labeling and confocal microscopy as well as coimmunoprecipitation analysis showed overlapping colocalization of polycystin-1 with alpha2beta1-integrin as well as with the focal adhesion proteins vinculin and paxillin in multiprotein clusters localized to focal areas of cell membrane contact with type I collagen matrix after short periods of attachment. Immunoprecipitation and Western immunoblot studies also showed that polycystin-1 was posttranslationally modified by tyrosine phosphorylation. These studies suggest that the PKD1-encoded protein is part of a large multiprotein complex in epithelial cells that functions in the regulation of extracellular matrix interactions with the plasma membrane and cell cytoskeleton.

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Cystic diseases of the kidney: role of adhesion molecules in normal and abnormal tubulogenesis.

Wilson PD, Burrow CR.

Mount Sinai School of Medicine, New York, N.Y., USA.
pat.wilson@SMTPlink.mssm.edu

This short review summarizes some information concerning what is known about matrix adhesion molecules, focal adhesion proteins, and cell-cell adhesion molecules in normal renal development and cystic diseases of the kidney. The focus is on human nephrogenesis and disease, but utilizes critical information gained from genetically manipulated mouse models. Interestingly, a significant role for the human PKD-1-encoded gene product, polycystin-1, has been found in cell-matrix interactions via integrins during development, and mutations lead to autosomal dominant polycystic kidney disease (ADPKD). Recent studies on human ADPKD have implicated polycystin-1 in the formation of multiprotein complexes containing focal adhesion proteins at the basal cell surface of the normal ureteric bud. Further evidence of a critical role of cell-matrix interactions via focal adhesion complex formation is provided by the development of renal cystic disease in tensin knockout mice.

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AUTHOR(S): Cho, Hyeseou (1); Kehrl, John H. (1)
CORPORATE SOURCE: (1) NIH, 10 Center Dr., Bldg. 10 Rm. 11B08, Bethesda, MD, 20892 USA
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A577. print.
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AB. . . contain additional domains known to interact with a variety of signaling molecules such as 14-3-3, Rap1/2, RhoA, Gbeta5, GIPC, and **polycystin**. RGS14, a larger member of the RGS family impairs Gialpha- and G13alpha-mediated signaling pathways and is strongly expressed in lymphocytes. To search for additional RGS14 functions, we performed a yeast 2-hybrid **screen** using a human spleen library with an RGS14 bait. We identified the human centrosome protein, ninein as well as several. . .

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DOCUMENT NUMBER: 20490726 PubMed ID: 10913159
TITLE: In vivo interaction of the adapter protein CD2-associated protein with the type 2 polycystic kidney disease protein, polycystin-2.
AUTHOR: Lehtonen S; Ora A; Olkkonen V M; Geng L; Zerial M; Somlo S;
Lehtonen E
CORPORATE SOURCE: Department of Pathology, Haartman Institute, University of Helsinki, P. O. Box 21, FIN-00014 Helsinki, Finland.
CONTRACT NUMBER: P50DK57328 (NIDDK)
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Last Updated on STN: 20010322
Entered Medline: 20001120
AB . . . at lower levels in renal tubular epithelial cells in the adult kidney, particularly in distal nephron segments. Independent yeast two-hybrid **screens** using the COOH-terminal region of either CD2AP or **polycystin**-2 as bait identified the COOH termini of **polycystin**-2 and CD2AP, respectively, as strong interacting partners. This interaction was confirmed in cultured cells by co-immunoprecipitation of endogenous **polycystin**-2 with endogenous CD2AP and vice versa. CD2AP shows a diffuse reticular cytoplasmic and perinuclear pattern of distribution, similar to **polycystin**-2, in cultured cells, and the two proteins co-localize by indirect double immunofluorescence microscopy. CD2AP is an adapter molecule that associates. . . membrane proteins to organize the cytoskeleton around a polarized site. Such a function fits well with that hypothesized for the **polycystin** proteins in renal tubular epithelial cells, and the present findings suggest that CD2AP has a role in **polycystin**-2 function.

L3 ANSWER 3 OF 4 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1998104524 MEDLINE
DOCUMENT NUMBER: 98104524 PubMed ID: 9442442
TITLE: Autosomal dominant polycystic kidney disease: clinical and genetic aspects.
AUTHOR: Sessa A; Ghiggeri G M; Turco A E
CORPORATE SOURCE: Department of Nephrology, G. Gaslini Children's Hospital, Genova, Italy.
SOURCE: JOURNAL OF NEPHROLOGY, (1997 Nov-Dec) 10 (6) 295-310.
Ref:
184
Journal code: CWE; 9012268. ISSN: 1120-3625.

PUB. COUNTRY: Italy
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980306
Last Updated on STN: 19980306
Entered Medline: 19980226

AB . . . studies on cystogenesis suggest a key role of cell-to-cell or cell-to-matrix interactions. Surface proteins mediating cell-to-cell contact, such as E-cadherin (**polycystin**?), integrin interactions, growth factors, receptor expression, are involved in the process of differentiation of the cellular condition and of the. . . (unknown chromosome) in a few families. PCR-based mutation detection methods, automated DNA sequencing, and other "functional" methods are used to **screen** and analyse ADPKD patients. It is not yet known whether the mutations identified so far in PKD1 and PKD2 inactivate the genes or generate an aberrant product. The products of PKD1 and PKD2 genes have been called **polycystin** 1 and 2. **Polycystins** are members of a family of interactive proteins involved in complex adhesive cell-cell, cell-matrix, protein-protein, and protein-carbohydrate interactions in the. . .

L3 ANSWER 4 OF 4 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 96108969 MEDLINE
DOCUMENT NUMBER: 96108969 PubMed ID: 8554072
TITLE: Screening the 3' region of the polycystic kidney disease 1 (PKD1) gene reveals six novel mutations.
AUTHOR: Peral B; San Millan J L; Ong A C; Gamble V; Ward C J; Strong C; Harris P C
CORPORATE SOURCE: MRC Molecular Haematology Unit, Institute of Molecular Medicine, John Radcliffe Hospital, Headington, Oxford, United Kingdom.
SOURCE: AMERICAN JOURNAL OF HUMAN GENETICS, (1996 Jan) 58 (1) 86-96.
Journal code: 3IM; 0370475. ISSN: 0002-9297.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199602
ENTRY DATE: Entered STN: 19960306
Last Updated on STN: 19960306
Entered Medline: 19960221

AB . . . kidney disease (ADPKD), PKD1 (polycystic kidney disease 1), has been fully characterized and shown to encode an integral membrane protein, **polycystin**, involved in cell-cell and/or cell-matrix interactions. Study of the PKD1 gene has been complicated because most of the gene lies. . . elsewhere on the same chromosome, and consequently only seven mutations have been described so far. Here we report a systematic **screen** covering approximately 80% of the approximately 2.75 kb of translated transcript that is encoded by single-copy DNA. We have identified. . . they indicate that the majority of mutations lie within the duplicated area, which is difficult to study. The regions of **polycystin** removed in each mutation so far described are assessed for their functional significance; an area disrupted by two new small. . .

=> s polycystin (p) atpase (p) collagen (p) focal (p) adhesion

L4 1 POLYCYSTIN (P) ATPASE (P) COLLAGEN (P) FOCAL (P) ADHESION

=> d l4 total kwic

L4 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2001 ACS
AB . . . to identify agents that regulate the activity of the polycystic kidney disease proteins encoded by the PKD-1 and PKD-2 genes (**polycystin**-1 and -2) and that may be useful in the treatment of polycystic kidney disease. The assays of the invention comprise. . . decrease in the PKD mediated mutant phenotype. Characteristics assocd. with such a mutant phenotype include increased adherence to type I **collagen**-coated surfaces; apical expression of NaK-**ATPase** on the cell membrane; increased expression of .beta.-2-NaK-**ATPase** ; and decreased **focal adhesion** kinase (FAK) incorporation into **focal adhesion** complexes, and inability to form tubular structures in a gel matrix. To facilitate the screening methods of the invention, cells. . . engineered to express epitope tagged PKD gene products and/or epitope tagged PKD interacting proteins (PKD-IP). Such interacting proteins include e.g. **focal adhesion** complex proteins such as FAK, paxillin, vinculin, and talin.

=> d l4 total ibib kwic

L4 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2001:507954 CAPLUS
TITLE: Polycystin-based screening methods for compounds useful in the treatment of polycystic kidney disease
INVENTOR(S): Wilson, Patricia D.; Burrow, Christopher R.
PATENT ASSIGNEE(S): Mount Sinai School of Medicine of New York University,
USA
SOURCE: PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001050130	A2	20010712	WO 2001-US100317	20010105

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-478737 A 20000106
US 2000-689461 A 20001012

AB Cell-based screening assays are provided which are designed to identify agents that regulate the activity of the polycystic kidney disease proteins encoded by the PKD-1 and PKD-2 genes (**polycystin**-1 and -2) and that may be useful in the treatment of polycystic kidney disease. The assays of the invention comprise the contacting of genetically engineered cells expressing a mutant or truncated PKD gene product with a test agent and assaying for a decrease in the PKD mediated mutant phenotype. Characteristics assocd. with such a mutant phenotype include

increased adherence to type I **collagen**-coated surfaces; apical expression of NaK-**ATPase** on the cell membrane; increased expression of .beta.-2-NaK-**ATPase**; and decreased **focal adhesion** kinase (FAK) incorporation into **focal adhesion** complexes, and inability to form tubular structures in a gel matrix. To facilitate the screening methods of the invention, cells may be genetically engineered to express epitope tagged PKD gene products and/or epitope tagged PKD interacting proteins (PKD-IP). Such interacting proteins include e.g. **focal adhesion** complex proteins such as FAK, paxillin, vinculin, and talin.

=> s polycystin (p) assay (p) expression

L5 11 POLYCYSTIN (P) ASSAY (P) EXPRESSION

=> dup rem l5

PROCESSING COMPLETED FOR L5
L6 4 DUP REM L5 (7 DUPLICATES REMOVED)

=> d l6 total ibib kwic

L6 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2001:507954 CAPLUS
TITLE: Polycystin-based screening methods for compounds useful in the treatment of polycystic kidney disease
INVENTOR(S): Wilson, Patricia D.; Burrow, Christopher R.
PATENT ASSIGNEE(S): Mount Sinai School of Medicine of New York University, USA
SOURCE: PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001050130	A2	20010712	WO 2001-US100317	20010105

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-478737 A 20000106
US 2000-689461 A 20001012

AB Cell-based screening **assays** are provided which are designed to identify agents that regulate the activity of the polycystic kidney disease proteins encoded by the PKD-1 and PKD-2 genes (**polycystin** -1 and -2) and that may be useful in the treatment of polycystic kidney disease. The **assays** of the invention comprise the contacting of genetically engineered cells expressing a mutant or truncated PKD gene product with a test agent and assaying for a decrease in the PKD mediated mutant phenotype. Characteristics assocd. with such a mutant phenotype include increased adherence to type I collagen-coated surfaces; apical **expression** of NaK-ATPase on the cell membrane; increased **expression** of .beta.-2-NaK-ATPase; and decreased focal adhesion kinase (FAK) incorporation into focal adhesion complexes, and inability to

form tubular structures in a gel matrix. To facilitate the screening methods of the invention, cells may be genetically engineered to express epitope tagged PKD gene products and/or epitope tagged PKD interacting proteins (PKD-IP). Such interacting proteins include e.g. focal adhesion complex proteins such as FAK, paxillin, vinculin, and talin.

L6 ANSWER 2 OF 4 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001017517 MEDLINE
DOCUMENT NUMBER: 20380163 PubMed ID: 10926175
TITLE: The pathogenesis of autosomal dominant polycystic kidney disease: an update.
AUTHOR: Somlo S; Markowitz G S
CORPORATE SOURCE: Department of Internal Medicine (Nephrology), Yale University School of Medicine, USA.
CONTRACT NUMBER: DK54053 (NIDDK)
DK57328 (NIDDK)
SOURCE: CURRENT OPINION IN NEPHROLOGY AND HYPERTENSION, (2000 Jul) 9 (4) 385-94. Ref: 69
Journal code: B4H. ISSN: 1062-4821.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001107

AB . . . normal allele in individual polarized epithelial cells. Most recent advances are focused on the function of the respective protein products, **polycystin**-1 and **polycystin**-2. Indirect evidence supports an interaction between **polycystin**-1 and -2, albeit it is unlikely that they work in concert in all tissues and at all times. They associate in yeast two hybrid and cotransfection **assays** and there is a striking similarity in the renal and pancreatic cystic phenotypes of Pkd2-/- and Pkd1del34/del34 mice. Also, the. . . human disease phenotypes remain completely overlapping with the major difference being in relative severity. Mounting evidence supports the hypothesis that **polycystin**-1 is a cell surface receptor. A close homologue in the sea urchin sperm mediates the acrosome reaction in response to. . . disease domains reveals a beta-sandwich fold commonly found in surface receptor molecules. Indirect evidence also supports the initial hypothesis that **polycystin**-2 is a calcium channel subunit. Several closely related homologues retain the calcium channel signature motif but differ in their predicted. . . shown to be a calcium regulated cation channel. Several important distinctions in polcystin-1 and -2 function have also been discovered. **Polycystin** -2 has a role in cardiac development that polcystin-1 does not. High level **polycystin**-2 **expression** in renal epithelial cells coincides with maturation and elongation of tubules and, unlike **polycystin**-1, persists into adulthood. In cells in tissue culture, **polycystin**-2 is expressed exclusively in the endoplasmic reticulum whilst the cellular **expression** of **polycystin**-1 remains unknown. Overall, the difficult task of understanding the autosomal dominant polycystic disease process is proceeding apace.

L6 ANSWER 3 OF 4 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999293063 MEDLINE
DOCUMENT NUMBER: 99293063 PubMed ID: 10362514
TITLE: Identification of phosphorylation sites in the PKD1-encoded protein C-terminal domain.
AUTHOR: Li H P; Geng L; Burrow C R; Wilson P D
CORPORATE SOURCE: Department of Medicine, Mount Sinai School of Medicine, New

York, New York 10029, USA.. Hsi-Ping_Li@mtplink.mssm.edu
CONTRACT NUMBER: RO1 DK448833 (NIDDK)
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1999 Jun 7) 259 (2) 356-63.
Journal code: 9Y8; 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990714
Last Updated on STN: 19990714
Entered Medline: 19990628

AB The PKD1-encoded protein, "**polycystin**-1", has a large N-terminal extracellular portion, multiple transmembrane domains, and a short intracellular C-terminal tail with four tyrosine residues and. . . in kidney development and autosomal dominant polycystic kidney disease (ADPKD) is still unknown. We have subcloned the cDNA encoding the **polycystin**-1 C-terminal domain (PKD1-CTD) into a prokaryotic **expression** vector, and site-directed mutagenesis was performed to target the four tyrosine residues and four serine residues in two putative phosphorylation sites. In vitro phosphorylation **assays** were conducted on both wild type and mutant PKD1-CTD fusion proteins. It was found that the wild type PKD1-CTD and. . .

L6 ANSWER 4 OF 4 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 96202312 MEDLINE
DOCUMENT NUMBER: 96202312 PubMed ID: 8643665
TITLE: Polycystin, the polycystic kidney disease 1 protein, is expressed by epithelial cells in fetal, adult, and polycystic kidney.
AUTHOR: Ward C J; Turley H; Ong A C; Comley M; Biddolph S; Chetty R; Ratcliffe P J; Gattner K; Harris P C
CORPORATE SOURCE: Medical Research Council Molecular Haematology Unit, Institute of Molecular Medicine, John Radcliffe Hospital, Headington, Oxford, United Kingdom.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1996 Feb 20) 93 (4) 1524-8.
Journal code: PV3; 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199607
ENTRY DATE: Entered STN: 19960726
Last Updated on STN: 19960726
Entered Medline: 19960717

AB . . . locus of the common genetic disorder autosomal dominant polycystic kidney disease. We have studied PKD1 mRNA, with an RNase protection **assay**, and found widespread **expression** in adult tissue, with high levels in brain and moderate signal in kidney. **Expression** of the PKD1 protein, **polycystin**, was assessed in kidney using monoclonal antibodies to a recombinant protein containing the C terminus of the molecule. In fetal and adult kidney, staining is restricted to epithelial cells. **Expression** in the developing nephron is most prominent in mature tubules, with lesser staining in Bowman's capsule and the proximal ureteric. . . persists in cortical tubules with moderate staining detected in the loops of Henle and collecting ducts. These results suggest that **polycystin**'s major role is in the maintenance of renal epithelial differentiation and organization from early fetal life. Interestingly, **polycystin expression**, monitored at the mRNA level and by immunohistochemistry, appears higher in cystic epithelia, indicating that the disease does not result. . .

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=> d his

     (FILE 'HOME' ENTERED AT 08:08:46 ON 31 JUL 2001)

     FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS, USPATFULL' ENTERED AT 08:08:58 ON
     31 JUL 2001
L1            0 S POLYCYSTIN (P) SCREEN (P) ATPASE (P) COLLAGEN (P) FOCAL (P)
A
L2           12 S POLYCYSTIN (P) SCREEN
L3            4 DUP REM L2 (8 DUPLICATES REMOVED)
L4            1 S POLYCYSTIN (P) ATPASE (P) COLLAGEN (P) FOCAL (P) ADHESION
L5           11 S POLYCYSTIN (P) ASSAY (P) EXPRESSION
L6            4 DUP REM L5 (7 DUPLICATES REMOVED)

=> s polycystin (p) assay (p) atpase (p) focal

L7            1 POLYCYSTIN (P) ASSAY (P) ATPASE (P) FOCAL

=> s polycystin (p) atpase (p) focal

L8            1 POLYCYSTIN (P) ATPASE (P) FOCAL

=> d l8 ibib


L8   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2001 ACS
ACCESSION NUMBER:         2001:507954  CAPLUS
TITLE:                    Polycystin-based screening methods for compounds
                          useful in the treatment of polycystic kidney disease
INVENTOR(S):              Wilson, Patricia D.; Burrow, Christopher R.
PATENT ASSIGNEE(S):       Mount Sinai School of Medicine of New York
University,
                          USA
SOURCE:                   PCT Int. Appl., 56 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.   DATE
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     WO 2001050130      A2   20010712       WO 2001-US100317 20010105
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
             HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
             LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
             SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU,
             ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
             DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
             BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.:                  US 2000-478737   A  20000106
                                        US 2000-689461   A  20001012


=> log y

COST IN U.S. DOLLARS                                  SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                      52.84      53.05

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)            SINCE FILE      TOTAL
                                                         ENTRY    SESSION
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STN INTERNATIONAL LOGOFF AT 08:16:08 ON 31 JUL 2001